

Hypertonic Saline Use in Acute Injury.

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Hypertonic solutions have been used in the resuscitation of different forms of shock since the late 1950s. Unlike isotonic solutions where the majority of infused fluid rapidly leaks out of the intravascular space, the hypertonic sodium gradient formed against the capillary membrane pulls in fluid from peripheral tissue allowing intravascular filling with a minute fluid infusion volume. Working very similarly to colloid resuscitation with albumin or starches, hypertonic solutions establish an osmotic gradient pulling free water from tissues into the circulation but without subsequent injury of capillary endothelium as can occur with colloid macromolecules.

In more recent years, hypertonic fluids have emerged as osmotherapy to reduce intracranial pressure in neurological emergencies such as traumatic brain injury, subarachnoid hemorrhage and stroke. The mechanism of action similarly involves the preferential dehydration of brain tissue moving brain water across the blood brain barrier into the circulation thereby shrinking swollen brain tissue and reducing intracranial pressure. To date only mannitol and hypertonic saline exist as the sole treatments beyond symptomatic relief in cases of intracranial hypertension leading to brain herniation and death.

Most interestingly though, is an additional effect of hypertonic solutions, discovered inadvertently in the last decade. A robust anti-inflammatory effect appears to be exerted by hypertonic saline on the innate immune system. In particular leukocyte and endothelial exposure to hypertonic solutions can blunt polymorphonuclear neutrophil (PMN) activation and its release of highly destructive proteinases and oxygen free radicals. Indiscriminate host activation of PMNs can occur with several systemic inflammatory conditions triggered by sepsis, injury, and pancreatitis and can lead to the multiorgan dysfunction syndrome (MODS) and death. Increasing evidence suggests that administration of hypertonic fluids before, during or after development of these conditions may prevent host progression to MODS; yet, to date no human trial has been able to demonstrate this.